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Modelling the Impact of Climate Changes on Schistosomiasis Epidemic in Human

Felix B. Mbogolela[1], **Estomih S. Massawe**[2]✉, **Daniel O. Makinde**[3]

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Author Affiliation:
[1]Department of Mathematics, University of Dar es Salaam, P. O. Box 35062, Dar es Salaam, Tanzania
[2]College of Education, Science and Mathematics, St. Joseph University in Tanzania, P. O. Box 11007, Dar es Salaam, Tanzania
[3]Faculty of Military Science, Stellenbosch University, Stellenbosch, Private Bag X2, Saldanha Bay, 7395, South Africa

✉**Corresponding Author:**
Estomih S. Massawe,
Email: emassawe2@gmail.com, estomihmassawe@yahoo.com

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ABSTRACT

In this paper, a deterministic model incorporating climate changes is formulated and analysed to study the impact of temperature factor and variation of river water levels on the Schistosomiasis disease transmission in human. Qualitative analysis is carried out to determine the basic reproduction number and equilibrium points. It is found that the disease free equilibrium is locally asymptotically stable if the reproduction number is less unity and endemic equilibrium point is globally asymptotically stable if the reproduction number is greater than unity. Numerical simulations are performed and suggest that at a certain range of temperature, i.e. 15.6° C to 30° C, the rate of humans and snails infection of the disease is high. On the other hand, the results show that the force of infection experienced by river water levels is high when the river water levels is low. The study therefore, recommends that treatments, good sanitation practices and human behavioural changes which reduce contact with water bodies should be practiced and applied in the community so as to reduce the infection intensity in the population.

Keywords: Climate Changes, Schistosomiasis, Epidemic in Human, Impact, Temperature

INTRODUCTION

Schistosomiasis, also known as snail fever or bilharzia, is a disease caused by parasitic flatworms called schistosomes (Colley *et al*., 2014). The urinary tract or the intestines may be infected. Symptoms include abdominal pain, diarrhoea, bloody stool, or blood in the urine. People are infected during routine agricultural, domestic, occupational, and recreational activities, which expose them to infested water. Lack of hygiene and certain play habits of school-aged children such as swimming or fishing in infested water make them vulnerable to infection.

In recent years, the impact of climate changes on human health has attracted considerable attention to many researchers. The effects on schistosomiasis epidemic diseases have been of an exceptional interest because of its disease burden and its transmission sensitivity to environmental conditions. The atmosphere, biosphere and hydrosphere are being perturbed in a manner which may prove irreversible (Martens *et al*., 1995). Different studies have shown that

the transmission potential of vector-borne diseases is very sensitive to climate changes. Schistosomiasis is the human infection probably with the most complex biological cycle, which involves at least two host species (human as a definitive host and snail as an intermediate host), two free living transmission stages of the parasite (cercariae and miracidia) and distinct environment (Yang, 2003).

Hu *et al*., (2015), investigated the spatio-temporal variations of S. japonicum infection risk in Anhui province and assessed the association of the disease with key environmental factors with the aim of understanding the mechanism of the disease and seeking clues to effective control of the disease. Their results suggested that seasonal variation of the normalized difference vegetation index, variation of land surface temperature at daytime and distance to the river were negatively significantly associated with the risk of schistosomiasis. So far not much has been done on how temperature factor and volume of river water affect the transmission dynamics of the disease in human. Therefore, in this paper, it is intended to formulate and analyse a mathematical model that determines the effects of temperature factors and variation of river water levels on the schistosomiasis disease transmission in human.

2. MODEL FORMULATION

In this section, a basic deterministic schistosomiasis model which explicitly incorporates environmental factors such as the effect of river water level where miracidia, cercariae and snails live and the effect of temperature in human is formulated and analysed. The model involves humans and snails populations respectively.

The model that is to be formulated is intended to analyse the impact of climate changes on the level of river water where cercariae, miracidia and snails live and the temperature factor on schistosomiasis transmission to humans. The model consists of six populations at any time t, namely: Susceptible humans $S_1(t)$, infected humans $I_1(t)$, treated humans $T(t)$, recovered humans $R(t)$, susceptible snails $S_2(t)$ and infected snails $I_2(t)$.

In this model, at any time t, new susceptible humans and snails recruits enter the populations through birth at constant rates of π_1 and π_2 respectively. There is a constant human natural death rate μ_1 and snails natural death rate μ_2. Infected humans and snails die due to disease at constant rates of m_1 and m_2 respectively. Similarly, infected humans recover from the disease or infection due to treatment at a rate of r_1 as well as natural recovery at a rate of γ. $N_1(t)$ is the total humans population given by

$$N_1(t) = S_1(t) + I_1(t) + T(t) + R(t). \tag{1}$$

Susceptible humans acquire schistosomiasis through infection by infected snails in the physical water environment at a rate of $\lambda_1(t)$ where

$$\lambda_1(t) = \frac{\beta_1(T) I_2(t)}{aS_1 + \in I_2(t)}, \tag{2}$$

with $\beta_1(T)$ being the rate of disease transmission to the susceptible humans by the infected snails in a river which is favoured by suitable environmental temperature T; $\in$ is the rate of disease transmission by infected snails due to water level and a is an impact of water level on the disease transmission.

On the other hand, $N_2(t)$ is the total intermediate host snails population given by

$$N_2(t) = S_2(t) + I_2(t). \tag{3}$$

Similarly, susceptible snails acquire schistosomiasis through infection by infected humans in the physical water environment at a rate of $\lambda_2(t)$ where

$$\lambda_2(t) = \frac{\beta_2(T) I_1(t)}{aS_2 + bI_1(t)}, \tag{4}$$

$\beta_2(t)$ being the rate of disease transmission to the susceptible snails by the infected human waste (urine or feces) in rivers which are favoured by suitable environmental temperature T, while b being the rate of disease transmission by infected human due to

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water level. The description of model state variables and parameters which are to be used in the model formulation are given in the table below.

Table 1: Description of State Variables and Parameters Used in the Model Formulation

Variable / Parameter	Description
$S_1(t)$	The number of susceptible humans at time t
$I_1(t)$	The number of infected humans at time t
$T(t)$	The number of treated individuals at time t
$R(t)$	The number of recovered individuals at time t
$S_2(t)$	The number of susceptible snails at time t
$I_2(t)$	The number of infected snails at time t
γ	Natural recovery rate of infected humans
$\beta_1(T)$	Disease transmission rate to the susceptible humans by the infected snails in the river
$\beta_2(T)$	Disease transmission rate to the susceptible snails by the infected human waste in the river
r_1	Recovery rate of infected humans due to treatment
π_1	Recruitment rate of susceptible humans
π_2	Recruitment rate of susceptible snails
μ_1	Human natural death rate
μ_2	Snail natural death rate
m_1	Human death rate due to disease
m_2	Snail death rate due to disease
$\in$	Rate of disease transmission by infected snails due to water level
b	Rate of disease transmission by infected humans due to water level
a	Impact of water level on disease transmission

In formulating the model, the following assumptions are taken into consideration:

1. The transmission of the disease in the human population is only through contact with infected snails in the physical water environment; Likewise the transmission of the disease in the snails population is only through contact with infected human waste in the physical water environment,
2. There is no vertical transmission of the disease,
3. There is no immigration of infectious humans in the population,
4. There is no immunity in both humans and snails populations,
5. Infected humans can recover naturally from the disease,
6. Infected humans recover from the infection due to treatment,
7. Variations of temperature due to climate changes affect snails population and contact patterns.

Taking into account the above considerations and assumptions, we have the following compartmental flow diagram:

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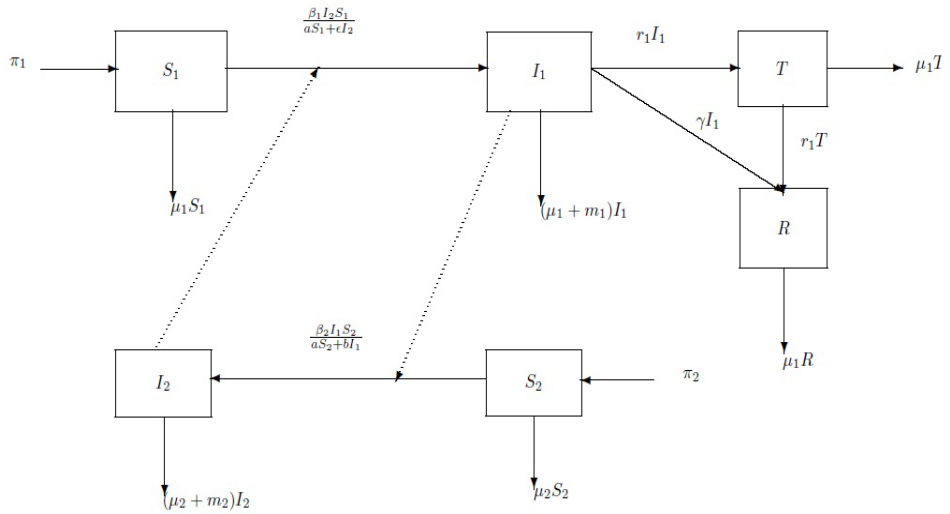

Figure 1: Compartmental Representation of the Model

From the above flow diagram, the dynamics of the disease is governed by the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS_1}{dt} &= \pi_1 - \lambda_1 S_1 - \mu_1 S_1 \\
\frac{dI_1}{dt} &= \lambda_1 S_1 - r_1 I_1 - \gamma I_1 - (m_1 + \mu_1) I_1 \\
\frac{dT}{dt} &= r_1 I_1 - \mu_1 T - r_1 T \\
\frac{dR}{dt} &= \gamma I_1 + r_1 T - \mu_1 R \\
\frac{dS_2}{dt} &= \pi_2 - \lambda_2 S_2 - \mu_2 S_2 \\
\frac{dI_2}{dt} &= \lambda_2 S_2 - (m_2 + \mu_2) I_2,
\end{aligned} \tag{5}$$

where λ_1 is a force of infection of the disease from the snails to humans, given by

$$\lambda_1 = \frac{\beta_1(T) I_2}{aS_1 + \in I_2}, \tag{6}$$

and λ_2 is also a force of infection of the disease from humans waste to snails, given by

$$\lambda_2 = \frac{\beta_2(T) I_1}{aS_2 + bI_1}. \tag{7}$$

3. MODEL ANALYSIS

In this section, the model system (5) will be qualitatively analysed to get insight to its disease transmission dynamics and understand the effect of climate changes on Schistosomiasis Epidemic in in humans. Threshold parameter which governs elimination or persistence of the disease in humans will be determined and analysed.

3.1. Positivity of Solutions

We prove that all solutions of the model system (5) with positive initial conditions remain positive for all the time $t > 0$. This is established by the following Lemma:

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Lemma 1 Let the initial data be $\{(S_1(0), I_1(0), T(0), R(0), S_2(0), I_2(0)) \geq 0\} \in \Omega$. Then, the solution set $\{S_1(t), I_1(t), T(t), R(t), S_2(t), I_2(t)\}$ of the model system (5) is positive for all $t \geq 0$.

Proof: From the first equation of the model system (5), we have

$$\frac{dS_1}{dt} = \pi_1 - \lambda_1 S_1 - \mu_1 S_1$$

or

$$\frac{dS_1}{dt} \geq -(\lambda_1 + \mu_1) S_1. \tag{8}$$

The inequality (8) has a solution

$$S_1(t) \geq S_1(0) e^{-\int_0^t (\lambda_1 + \mu_1) dt} \geq 0,$$

where

$$\lambda_1 = \frac{\beta_1(T) I_2}{a S_1 + \in I_2}.$$

Similarly, it can be shown that the remaining equations of the model system (5) have positive solutions.

Hence as $t \to \infty$, the solution set $\{S_1(t), I_1(t), T(t), R(t), S_2(t), I_2(t)\}$ of the model system (5) is positive for all $t \geq 0$.

3.2. Invariant Region

Since the model (5) is Schistosomiasis model dealing with humans and snails populations, we assume that all state variables and parameters of the model are positive for all $t \geq 0$. The model (5) will then be analysed in suitable feasible region where all state variables are uniformly bounded in a proper subset $\Omega \subseteq \mathbb{R}^6_+$.

The total population sizes of human N_1 and snails N_2 are given by

$$N_1 = S_1 + I_1 + T + R$$

and

$$N_2 = S_2 + I_2.$$

respectively.

From the differential equations of the total human population, we have

$$\begin{aligned} \frac{dN_1}{dt} &= \frac{dS_1}{dt} + \frac{dI_1}{dt} + \frac{dT}{dt} + \frac{dR}{dt}, \\ &= \pi_1 - (S_1 + I_1 + T + R)\mu_1 - m_1 I_1, \\ &= \pi_1 - N_1 \mu_1 - m_1 I_1. \end{aligned} \tag{9}$$

It is noted that in the absence of schistosomiasis, there are no deaths of humans due to the disease, that is $m_1 = 0$. Consequently, equation (9) becomes

$$\frac{dN_1}{dt} \leq \pi_1 - N_1 \mu_1. \tag{10}$$

The inequality (10) has a solution of the form

$$N_1(t) = \frac{\pi_1}{\mu_1} - \left(\frac{\pi_1 - N_1(0)\mu_1}{\mu_1} \right) e^{-\mu_1 t}. \quad (11)$$

As $t \to \infty$, the population size $N_1(t)$ approaches $\frac{\pi_1}{\mu_1}$.

Similarly, for the population of snails we have

$$\frac{dN_2}{dt} = \frac{dS_2}{dt} + \frac{dI_2}{dt}, \quad (12)$$

$$= \pi_2 - (S_2 + I_2)\mu_2 - m_2 I_2,$$

$$= \pi_2 - N_2\mu_2 - m_2 I_2. \quad (13)$$

In the absence of schistosomiasis disease, there are no deaths of the snails from the disease, that is $m_2 = 0$, implying that

$$\frac{dN_2}{dt} \le \pi_2 - N_2\mu_2. \quad (14)$$

The inequality (14) has a solution of the form

$$N_2(t) \le \frac{\pi_2}{\mu_2} - \left(\frac{\pi_2 - N_2(0)\mu_2}{\mu_2} \right) e^{-\mu_2 t}. \quad (15)$$

As $t \to \infty$, the population size $N_2(t)$ approaches $\frac{\pi_2}{\mu_2}$.

It can be seen that, the humans and snails populations tend to zero as $t \to \infty$. It is therefore concluded that, all feasible solutions of the model system (5) are positive and eventually enter the invariant attracting region

$$\Omega = \left\{ (S_1, I_1, T, R, S_2, I_2) \in \mathbb{R}_+^6 : 0 \le N_1 \le \frac{\pi_1}{\mu_1}, 0 \le N_2 \le \frac{\pi_2}{\mu_2} \right\}. \quad (16)$$

Thus, whenever $\pi_1 \ge \mu_1$ and $\pi_2 \ge \mu_2$, Ω is positively invariant and attracting. Then the basic mathematical model is well posed and it is mathematically relevant, therefore it is sufficient to study the dynamics of the epidemiological system (5) in the region Ω.

3.3. Existence and Stability of Equilibrium Points

In this section, the existence of equilibrium points of the model system (5) and their stabilities are determined.

Let $E(S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*)$ be the equilibrium points of the model system (5). Then the equilibrium points of the model system are obtained by setting the right hand side of the model system (5) equal to zero, that is:

$$\begin{aligned}
0 &= \pi_1 - \lambda_1 S_1^* - \mu_1 S_1^* \\
0 &= \lambda_1 S_1^* - r_1 I_1^* - \gamma I_1^* - (m_1 + \mu_1) I_1^* \\
0 &= r_1 I_1^* - \mu_1 T^* - r_1 T^* \\
0 &= \gamma I_1^* + r_1 T^* - \mu_1 R^* \\
0 &= \pi_2 - \lambda_2 S_2^* - \mu_2 S_2^* \\
0 &= \lambda_2 S_2^* - (m_2 + \mu_2) I_2^*
\end{aligned} \quad (17)$$

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Note that, the population of both humans and snails will never be extinct as long as humans and snails recruitment terms π_1 and π_2 are not zero respectively. This implies that there is no trivial equilibrium point.

3.4. Disease Free-Equilibrium Point, E_0

Disease free equilibrium point (DFE), is a steady state solution of the system when there is no disease. Therefore, in the absence of infection, that is, when $I_1^* = I_2^* = 0$, the model system (17) will reduce to

$$0 = \pi_1 - \mu_1 S_1$$

which implies that

$$S_1^* = \frac{\pi_1}{\mu_1}. \tag{18}$$

Similarly

$$0 = -(\mu_1 + r_1)T,$$

that is,

$$T^* = 0. \tag{19}$$

Also

$$0 = r_1 T - \mu_1 R.$$

But $T = 0$; therefore $R^* = 0$.

Furthermore,

$$0 = \pi_2 - \mu_2 S_2,$$

that is

$$S_2^* = \frac{\pi_2}{\mu_2}. \tag{20}$$

Thus, from the equations (17), the disease free equilibrium point of the model is given by

$$E_0 = \left(S_1^*, 0,\ T^*, R^*, S_2^*, 0\right) = \left[\frac{\pi_1}{\mu_1},\ 0,\ 0,\ 0,\ \frac{\pi_2}{\mu_2},\ 0\right]. \tag{21}$$

3.5. The Basic Reproduction Number, R_0

Diekmann *et al*., (1990), defined the basic reproduction number R_0, as the average number of secondary infections produced by a single infectious host, during his or her entire period of infectiousness. The basic reproduction number is one of the most important non-dimensional tool in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies.

The basic reproduction number R_0, is computed by using the next generation operator method (Van den Driessche and Watmough, 2002) as follows:

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Let

(i) $F_i(x)$ be the rate of appearance of new infection in compartment i,

(ii) $V_i^+(x)$ be the transfer of individuals into compartment i by all other means,

(iii) $V_i^-(x)$ be the rate of transfer of individuals out of compartment i where it is assumed that each of the functions F_i, V_i^+ and V_i^- are continuously differentiable at least twice with respect to each variable and $V_i = V_i^- - V_i^+$,

(iv) X_0 be the disease free equilibrium point.

According to Diekmann and Heestereek (2000), we call $\mathbf{FV}^-$ the next generation matrix for the model and set the reproduction number equal to

$$R_0 = \rho\left(\mathbf{FV}^-\right)$$

where

$$\mathbf{F} = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] \text{ and } \mathbf{V} = \left[\frac{\partial V_i(x_0)}{\partial x_j}\right]^{-1}$$

with $i \geq 1$, for the number of compartments and $1 \leq j \leq m$ for the infected compartments only. $\rho(\mathbf{A})$ denotes the spectral radius of a matrix $\mathbf{A}$. $\mathbf{F}$ and $\mathbf{V}$ are $m \times m$ matrices, where m is the number of infected classes. Consider an infected individual introduced into compartment k of a disease-free population. The $(i, j)^{th}$ entry of $\mathbf{F}$ is the rate at which an infected individual in compartment j produces new infections in compartment i, and the $(j,k)^{th}$ entry of $\mathbf{V}^{-1}$ is the average time an infected individual spends in compartments j during its lifetime in compartment k. Hence, the $(i,k)^{th}$ entry of the product $\mathbf{FV}^{-1}$ is the expected number of new infections in compartment i produced by the infected individual originally introduced into compartment k (Van den Driessche and Watmough, 2002). We re-write the system (5) as

$$\begin{aligned}
\frac{dI_1}{dt} &= \lambda_1 S_1 - r_1 I_1 - \gamma I_1 - (m_1 + \mu_1) I_1, \\
\frac{dI_2}{dt} &= \lambda_2 S_2 - (m_2 + \mu_2) I_2, \\
\frac{dT}{dt} &= r_1 I_1 - \mu_1 T - r_1 T, \\
\frac{dR}{dt} &= \gamma I_1 + r_1 T - \mu_1 R, \\
\frac{dS_1}{dt} &= \pi_1 - \lambda_1 S_1 - \mu_1 S_1, \\
\frac{dS_2}{dt} &= \pi_2 - \lambda_2 S_2 - \mu_2 S_2.
\end{aligned} \tag{22}$$

Following Van den Driessche and Watmough (2002), we define $\mathbf{F}_i$ and $\mathbf{V}_i$ from the system (22) as

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$$\mathbf{F_i} = \begin{bmatrix} \dfrac{\beta_1(T) S_1 I_2}{aS_1 + \in I_2} \\ \dfrac{\beta_2(T) S_2 I_1}{aS_2 + bI_1} \end{bmatrix}, \tag{23}$$

and

$$\mathbf{V}_i = \begin{bmatrix} r_1 I_1 + \gamma I_1 + (m_1 + \mu_1) I_1 \\ (m_2 + \mu_2) I_2 \end{bmatrix}. \tag{24}$$

Now the partial derivatives of (23) with respect to I_1 and I_2 gives

$$D\mathbf{F}_i = \begin{bmatrix} 0 & \dfrac{a\beta_1(T) S_1^2}{(aS_1 + \in I_2)^2} \\ \dfrac{a\beta_2 S_2^2}{(aS_2 + bI_2)^2} & 0 \end{bmatrix}. \tag{25}$$

At disease-free equilibrium point E_0,

$$\mathbf{F} = \begin{bmatrix} 0 & \dfrac{\beta_1(T)}{a} \\ \dfrac{\beta_2(T)}{a} & 0 \end{bmatrix}. \tag{26}$$

Similarly, the partial differentiation of (24) yields

$$D\mathbf{V_i} = \begin{bmatrix} (r_1 + m_1 + \mu_1 + \gamma) & 0 \\ 0 & (m_2 + \mu_2) \end{bmatrix},$$

which when evaluated a E_0 gives

$$D\mathbf{V_i}(x_0) = \begin{bmatrix} (r_1 + m_1 + \mu_1 + \gamma) & 0 \\ 0 & (m_2 + \mu_2) \end{bmatrix}. \tag{27}$$

Note that $\mathbf{V} = D\mathbf{V}_i(x_0)$. Now when we take the inverse of matrix (27) we get

$$\mathbf{V^{-1}} = \begin{bmatrix} \dfrac{1}{m_1 + r_1 + \mu_1 + \gamma} & 0 \\ 0 & \dfrac{1}{m_2 + \mu_2} \end{bmatrix}. \tag{28}$$

Consequently,

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$$\mathbf{FV}^{-1}=\begin{bmatrix} 0 & \dfrac{\beta_1(T)}{a(m_2+\mu_2)} \\ \dfrac{\beta_2(T)}{(\gamma+m_1+r_1+\mu_1)} & 0 \end{bmatrix}. \tag{29}$$

The basic reproduction number, R_0 is defined as the spectral radius (dominant eigenvalue) of the matrix $\mathbf{FV}^{-1}$ i.e., $R_0=\rho\left(\mathbf{FV}^{-1}\right)$ is determined by calculating the eigenvalues of the matrix (29) above. The calculated eigenvalues of matrix (29) above are given by

$$\lambda_1=-\frac{\sqrt{\beta_1(T)\beta_2(T)}}{a\sqrt{(\gamma+\mu_1+r_1+m_1)(m_2+\mu_2)}}$$

and

$$\lambda_2=\frac{\sqrt{\beta_1(T)\beta_2(T)}}{a\sqrt{(\gamma+\mu_1+r_1+m_1)(m_2+\mu_2)}}$$

It is clearly seen that λ_2 is the dominant eigenvalue, hence it is the reproduction number of the model. Therefore,

$$R_0=\frac{\sqrt{\beta_1(T)\beta_2(T)}}{a\sqrt{(\gamma+\mu_1+r_1+m_1)(m_2+\mu_2)}}. \tag{30}$$

3.6. Local Stability of the Disease Free-Equilibrium Point

The local stability of the disease-free equilibrium point $E_0=\left[\frac{\pi_1}{\mu_1},\ 0,\ 0,\ 0,\ \frac{\pi_2}{\mu_2},\ 0\right]$ is examined by the linearized form of the model system (5) at the given steady state E_0. We linearize the model system (5) by computing its Jacobian matrix $\mathbf{J}_E$. We re-define the system (5) as

$$\begin{aligned}
f_1(S_1,I_1,T,R,S_2,I_2)&=\pi_1-\lambda_1S_1-\mu_1S_1,\\
f_2(S_1,I_1,T,R,S_2,I_2)&=\lambda_1S_1-r_1I_1-\gamma I_1-(m_1+\mu_1)I_1,\\
f_3(S_1,I_1,T,R,S_2,I_2)&=r_1I_1-\mu_1T-r_1T,\\
f_4(S_1,I_1,T,R,S_2,I_2)&=r_1I_1+r_1T-\mu_1R,\\
f_5(S_1,I_1,T,R,S_2,I_2)&=\pi_2-\lambda_2S_2-\mu_2S_2,\\
f_6(S_1,I_1,T,R,S_2,I_2)&=\lambda_2S_2-(\mu_2+m_2)I_2.
\end{aligned} \tag{31}$$

Hence, at the steady states, the Jacobian matrix of $f_1,\ f_2,\ f_3,\ f_4,\ f_5$ and f_6 with respect to S_1, I_1, T, R, S_2 and I_2 is given by

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$$\mathbf{J}_E = \begin{bmatrix} \frac{\partial f_1}{\partial S_1}(E_0) & \frac{\partial f_1}{\partial I_1}(E_0) & \frac{\partial f_1}{\partial T}(E_0) & \frac{\partial f_1}{\partial R}(E_0) & \frac{\partial f_1}{\partial S_2}(E_0) & \frac{\partial f_1}{\partial I_2}(E_0) \\ \frac{\partial f_2}{\partial S_1}(E_0) & \frac{\partial f_2}{\partial I_1}(E_0) & \frac{\partial f_2}{\partial T}(E_0) & \frac{\partial f_2}{\partial R}(E_0) & \frac{\partial f_2}{\partial S_2}(E_0) & \frac{\partial f_2}{\partial I_2}(E_0) \\ \frac{\partial f_3}{\partial S_1}(E_0) & \frac{\partial f_3}{\partial I_1}(E_0) & \frac{\partial f_3}{\partial T}(E_0) & \frac{\partial f_3}{\partial R}(E_0) & \frac{\partial f_3}{\partial S_2}(E_0) & \frac{\partial f_3}{\partial I_1}(E_0) \\ \frac{\partial f_4}{\partial S_1}(E_0) & \frac{\partial f_4}{\partial I_1}(E_0) & \frac{\partial f_4}{\partial T}(E_0) & \frac{\partial f_4}{\partial R}(E_0) & \frac{\partial f_4}{\partial S_2}(E_0) & \frac{\partial f_4}{\partial I_1}(E_0) \\ \frac{\partial f_5}{\partial S_1}(E_0) & \frac{\partial f_5}{\partial I_1}(E_0) & \frac{\partial f_5}{\partial T}(E_0) & \frac{\partial f_5}{\partial R}(E_0) & \frac{\partial f_5}{\partial S_2}(E_0) & \frac{\partial f_5}{\partial I_2}(E_0) \\ \frac{\partial f_6}{\partial S_1}(E_0) & \frac{\partial f_6}{\partial I_1}(E_0) & \frac{\partial f_6}{\partial T}(E_0) & \frac{\partial f_6}{\partial R}(E_0) & \frac{\partial f_6}{\partial S_2}(E_0) & \frac{\partial f_6}{\partial I_1}(E_0) \end{bmatrix},$$

which turns out to be

$$\mathbf{J}_E = \begin{bmatrix} -(F_{11}+\mu_1) & 0 & 0 & 0 & 0 & -F_{16} \\ F_{21} & -F_{22} & 0 & 0 & 0 & F_{26} \\ 0 & r_1 & -(\mu_1+r_1) & 0 & 0 & 0 \\ 0 & \gamma & r_1 & -\mu_1 & 0 & 0 \\ 0 & -F_{52} & 0 & 0 & -(F_{55}+\mu_2) & 0 \\ 0 & F_{62} & 0 & 0 & F_{65} & -(m_2+\mu_2) \end{bmatrix} \quad (32)$$

where

$$F_{11} = \left[\frac{\beta_1 \in I_2^2}{(aS_1 + \in I_2)^2}\right], \quad F_{16} = \left[\frac{\beta_1 a S_1^2}{(aS_1 + \in I_2)^2}\right], \quad F_{21} = \left[\frac{\beta_1 \in I_2^2}{(aS_1 + I_2)^2}\right], \quad F_{22} = \left[r_1 + m_1 + \mu_1 + \gamma\right],$$

$$F_{26} = \left[\frac{\beta_1 a S_1^2}{(aS_1 + \in I_2)^2}\right], \; F_{52} = \left[-\frac{\beta_2 a S_2^2}{(aS_2 + bI_2)^2}\right], \; F_{55} = \left[-\frac{\beta_2 b I_1^2}{(aS_2 + bI_1)^2}\right], \; F_{62} = \left[\frac{\beta_2 a S_2^2}{(aS_2 + bI_2)^2}\right]$$

and $F_{65} = \left[\frac{\beta_2 b I_1^2}{(aS_2 + bI_1)^2}\right]$.

Now, when we substitute the values of E_0 in the matrix (32) above, at disease-free equilibrium point the matrix becomes:

$$\mathbf{J}_{E_0} = \begin{bmatrix} -\mu_1 & 0 & 0 & 0 & 0 & -\frac{\beta_1}{a} \\ 0 & -L_{22} & 0 & 0 & 0 & \frac{\beta_1}{a} \\ 0 & r_1 & -(\mu_1+r_1) & 0 & 0 & 0 \\ 0 & \gamma & r_1 & -\mu_1 & 0 & 0 \\ 0 & -\frac{\beta_2}{A} & 0 & 0 & -\mu_2 & 0 \\ 0 & \frac{\beta_2}{a} & 0 & 0 & 0 & -(m_2+\mu_2) \end{bmatrix}, \quad (33)$$

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where

$$L_{22} = \left[\left(r_1 + m_1 + \mu_1 + \gamma\right)\right].$$

We compute the eigenvalues λ of the matrix (33) above from the determinant $\left|\mathbf{J}_{E_0}\right| = 0$ where

$$\mathbf{J}_{E_0} = \begin{bmatrix} \mu_1 - \lambda & 0 & 0 & 0 & 0 & -\frac{\beta_1}{a} \\ 0 & L_{22} - \lambda & 0 & 0 & 0 & \frac{\beta_1}{a} \\ 0 & r_1 & -L_{33} - \lambda & 0 & 0 & 0 \\ 0 & \gamma & r_1 & -\mu_1 - \lambda & 0 & 0 \\ 0 & -\frac{\beta_2}{a} & 0 & 0 & \mu_2 - \lambda & 0 \\ 0 & \frac{\beta_2}{a} & 0 & 0 & 0 & -L_{66} - \lambda \end{bmatrix} \tag{34}$$

with

$$L_{22} = \left[\left(r_1 + m_1 + \mu_1 + \gamma\right)\right],\ L_{33} = \left(\mu_1 + r_1\right), \text{ and } L_{66} = \left(m_2 + \mu_2\right).$$

From the matrix (34) above, it is clearly seen that there are three eigenvalues which have negative real parts i.e., $\lambda_1 = -\mu_1$, $\lambda_2 = -\mu_1$, and $\lambda_3 = -\mu_2$. The remaining three eigenvalues are obtained from the determinant

$$\begin{vmatrix} -\left(r_1 + m_1 + \mu_1 + \gamma\right) - \lambda & 0 & \frac{\beta_1}{a} \\ r_1 & -\left(m_1 + r_1\right) - \lambda & 0 \\ \frac{\beta_2}{a} & 0 & -\left(m_2 + \mu_2\right) - \lambda \end{vmatrix} = 0. \tag{35}$$

This gives

$$\left(\lambda + \gamma + m_1 + r_1 + \mu_2\right)\left(\lambda + m_2 + \mu_2\right) - \frac{\beta_1\beta_2}{a^2} = 0. \tag{36}$$

If we let

$$A_1 = \left(\gamma + m_1 + r_1 + \mu_1\right),$$
$$A_2 = \left(m_2 + \mu_2\right),$$

and

$$C = \frac{\beta_1\beta_2}{a^2}.$$

We can write (36) as

$$\left(A_1 + \lambda\right)\left(A_2 + \lambda\right) - C = 0. \tag{37}$$

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Thus

$$\lambda^2 + (A_1 + A_2)\lambda + A_1A_2 - C = 0. \tag{38}$$

According to Routh-Hurwitz criteria, E_0 is locally asymptotically stable if $A_1 + A_2 > 0$ and $A_1A_2 - C > 0.$

Then it can be shown that

$$A_1 + A_2 = (m_1 + r_1 + \mu_1 + \gamma) + (m_2 + \mu_2) > 0, \tag{39}$$

and

$$AA_2 - C = (m_1 + r_1 + \mu_1 + \gamma)(m_2 + \mu_2) - \frac{\beta_1\beta_2}{a^2} > 0. \tag{40}$$

Thus

$$\frac{\beta_1\beta_2}{a^2} < (m_1 + r_1 + \mu_1 + \gamma)(m_2 + \mu_2),$$

or

$$\frac{\beta_1\beta_2}{a^2(m_1 + r_1 + \mu_1 + \gamma)(m_2 + \mu_2)} < 1. \tag{41}$$

This yields

$$\frac{\sqrt{\beta_1\beta_2}}{a\sqrt{(m_1 + r_1 + \mu_1 + \gamma)(m_2 + \mu_2)}} < 1,$$

implying that

$$R_0 = \frac{\sqrt{\beta_1(T)\beta_2(T)}}{\sqrt{(m_1 + r_1 + \mu_1 + \gamma)(m_2 + \mu_2)}} < 1. \tag{42}$$

Thus, E_0 is locally asymptotically stable if $R_0 < 1$.

3.7. The Endemic Equilibrium State and its Stability

Endemic equilibrium point E_1, is a steady state solution when the disease persist in the population. At the endemic equilibrium state, both humans and snails in the population are infected by schistosomiasis, and note that the endemic equilibrium is given by

$$E^* = (S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*). \tag{43}$$

By considering the model system (5), the endemic equilibrium value for susceptible humans is given by

$$S_1^* = \frac{\pi_1}{\mu_1 + \lambda_1^*}. \tag{44}$$

The endemic equilibrium value for infected humans with schistosomiasis is given by

$$I_1^* = \frac{\pi_1\lambda_1^*}{(\lambda_1^* + \mu_1)(m_1 + \mu_1 + r_1 + \gamma)}. \tag{45}$$

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Likewise the endemic equilibrium point for individuals getting treatment is given by

$$T^* = \frac{\pi_1 r_1 \lambda_1^*}{\left(\lambda_1^* + \mu_1\right)\left(m_1 + \mu_1 + r_1 + \gamma\right)\left(\mu_1 + r_1\right)}. \tag{46}$$

In the case of recovered class, the endemic equilibrium point is given by

$$R^* = \frac{\pi_1 \lambda_1^* \left(\gamma\mu_1 + \gamma r_1 + r_1^2\right)}{\left(\lambda_1^* + \mu_1\right)\left(m_1 + \mu_1 + r_1 + \gamma\right)\left(\mu_1 + r_1\right)\mu_1}. \tag{47}$$

In case of snails, the endemic equilibrium value for susceptible snails in the population is given by

$$S_2^* = \frac{\pi_2}{\mu_2 + \lambda_2^*}. \tag{48}$$

Likewise the endemic equilibrium value for infected snails is given by

$$I_2^* = \frac{\lambda_2^* S_2^*}{m_2 + \mu_2} = \frac{\pi_2 \lambda_2^*}{\left(\mu_2 + \lambda_2^*\right)\left(m_2 + \mu_2\right)}. \tag{49}$$

3.7.1. The Existence of the Endemic Equilibrium Point, E_1

In the presence of schistosomiasis disease, that is $I_1 \neq 0$ and $I_2 \neq 0$, the model system (5), has an endemic equilibrium point given by

$$E_1 = \left(S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*\right) \neq 0, \tag{50}$$

where

$\left(S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*\right) > 0$ and $S_1' = I_1' = T' = R' = S_2' = I_2' = 0$.

Let $E^* = \left(S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*\right)$ be a constant solution of the model system (5). We then express $E^* = \left(S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*\right)$ in terms of I_2^* as

$$\begin{aligned}
S_1^*\left(I_2^*\right) &= \frac{\pi_1\left(a + \in I_2\right)}{\mu_1\left(a + \in I_2\right) + \beta_1 I_2}, \\
I_1^*\left(I_2^*\right) &= \frac{\pi_1 \beta_1 I_2}{\left(r_1 + \mu_1\right)\left(\gamma + m_1 + r_1 + \mu_1\right)\left(I_2\beta_1 + \left(a + \in I_2\right)\mu_1\right)}, \\
T^*\left(I_2^*\right) &= \frac{\pi_1 r_1 \beta_1 I_2^*}{\left(r_1 + \mu_1\right)\left(\gamma + m_1 + r_1 + \mu_1\right)\left(I_2\beta_1 + \left(a + \in I_2\right)\mu_1\right)}, \\
R^*\left(I_2^*\right) &= \frac{\pi_1 \beta_1 \left(r_1^2 + \gamma r_1 + \mu_1\gamma\right) I_2}{\left(r_1 + \mu_1\right)\left(\gamma + m_1 + r_1 + \mu_1\right)\left(I_2\beta_1 + \left(a + \in I_2\right)\mu_1\right)}, \\
S^*\left(I_2^*\right) &= \frac{\pi_2\left(a + bA\right)}{\beta_2 A + \mu_2\left(a + bA\right)},
\end{aligned} \tag{51}$$

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$$\lambda_1^*\left(I_2^*\right)=\frac{\beta_1 I_2}{a+\in I_2},$$

$$\lambda_2^*\left(I_2^*\right)=\frac{\pi_1\beta_1\beta_2}{aA+b\pi_1\beta_1 I_2},$$

where

$$A=\left(r_1+\mu_1\right)\left(\gamma+m_1+r_1+\mu_1\right)\left(I_2\beta_1+\left(a+\in I_2\right)\mu_1\right).$$

Now we substitute the expression (51) for I_2 which is given by

$$\frac{dI_2}{dt}=\lambda_2 S_2-\left(m_2+\mu_2\right)I_2. \tag{52}$$

at the endemic equilibrium we get

$$I_2\left[\frac{\pi_1\pi_2\beta_1\beta_2}{\pi_1\beta_1\beta_2 I_2+a\mu_2 A+\mu_2 bI_2\pi_1\beta_1}-\left(m_2+\mu_2\right)\right]=0. \tag{53}$$

where

$$A=\left(r_1+\mu_1\right)\left(\gamma+m_1+r_1+\mu_1\right)\left(I_2\beta_1+\left(a+\in I_2\right)\mu_1\right).$$

Equation (53) gives $I_2^*=0$ which corresponds to the disease free-equilibrium point and at the endemic equilibrium point it can be shown that $R_0>1$ by considering the same expression (53) with some assumptions. By considering the equation

$$I_2\left[\frac{\pi_1\pi_2\beta_1\beta_2}{\pi_1\beta_1\beta_2 I_2+a\mu_2 A+\mu_2 bI_2\pi_1\beta_1}-\left(m_2+\mu_2\right)\right]=0$$

at disease free-equilibrium point where $I_2=0$ we obtain

$$\frac{\pi_1\pi_2\beta_1\beta_2}{a^2\left(\mu_1+r_1\right)\left(m_1+r_1+\mu_1+\gamma\right)\mu_1\mu_2}-\left(m_2+\mu_2\right)=0$$

$$\frac{\beta_1\beta_2}{a^2\left(\mu_1+r_1\right)\left(m_2+\mu_2+\mu_1+\gamma\right)}=\frac{\mu_1\mu_2\left(\mu_1+r_1\right)}{\pi_1\pi_2}>1 \tag{54}$$

Thus

$$\frac{\beta_1\beta_2}{a^2\left(\mu_1+r_1\right)\left(m_2+\mu_2+\mu_1+\gamma\right)}>1.$$

We then have the following expression which belong to the existence of the disease at endemic equilibrium:

$$R_0=\frac{1}{a}\sqrt{\frac{\beta_1\beta_2}{\left(\mu_1+r_1\right)\left(m_2+\mu_2+\mu_1+\gamma\right)}}>0. \tag{55}$$

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We can only deduce from expression (55) that the only one positive endemic equilibrium exists for $R_0 > 0$. Consequently, there exist one unique endemic equilibrium for model system (5) whenever $R_0 > 0$.

3.7.2. Global stability of the endemic equilibrium point, E_1

Theorem 4: If $R_0 > 1$, the endemic equilibrium E_1 of the model system (5) is globally asymptotically stable otherwise it is unstable.

Proof:

We develop the global stability of the endemic equilibrium E_1 by forming the following Lyapunov function: $V\left(S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*\right)$ defined by

$$V = \left(S_1 - S_1^* - S_1^* \ln \frac{S_1^*}{S_1}\right) + \left(I_1 - I_1^* - I_1^* \ln \frac{I_1^*}{I_1}\right) + \left(T - T^* - T^* \ln \frac{T^*}{T}\right) + \left(R - R^* - R^* \ln \frac{R^*}{R}\right) + \left(S_2 - S_2^* - S_2^* \ln \frac{S_2^*}{S_2}\right) + \left(I_2 - I_2^* - I_2^* \ln \frac{I_2^*}{I_2}\right).$$

Consequently,

$$\frac{dV}{dt} = \left(\frac{S_1 - S_1^*}{S_1}\right)\frac{dS_1}{dt} + \left(\frac{I_1 - I_1^*}{I_1}\right)\frac{dI_1}{dt} + \left(\frac{T - T^*}{T}\right)\frac{dT}{dt} + \left(\frac{R - R^*}{R}\right)\frac{dR}{dt} + \left(\frac{S_2 - S_2^*}{S_2}\right)\frac{dS_2}{dt} + \left(\frac{I_2 - I_2^*}{I_2}\right)\frac{dI_2}{dt}. \quad (56)$$

Now, when we substitute the expressions for $\frac{dS_1}{dt}, \frac{dI_1}{dt}, \frac{dT}{dt}, \frac{dR}{dt}, \frac{dS_2}{dt}, \frac{dI_2}{dt}$ from the model system (5) to obtain

$$\begin{aligned}\frac{dV}{dt} = {} & \left(\frac{S_1 - S_1^*}{S_1}\right)\left[\pi_1 - \frac{\beta_1 S_1 I_2}{aS_1 + \in I_2} - \mu_1 S_1\right] \\ & + \left(\frac{I_1 - I_1^*}{I_1}\right)\left[\frac{\beta_1 S_1 I_2}{aS_1 + \in I_2} - (r_1 + m_1 + \mu_1 + \gamma) I_1\right]. \\ & + \left(\frac{T - T^*}{T}\right)\left[r_1 I_1 - (\mu_1 + \gamma) T\right] \\ & + \left(\frac{R - R^*}{R}\right)\left[\gamma I_1 + r_1 T - \mu_1 R\right] \\ & + \left(\frac{S_2 - S_2^*}{S_2}\right)\left[\pi_2 - \frac{\beta_2 S_2 I_1}{aS_2 + bI_1} - \mu_2 S_2\right] \\ & + \left(\frac{I_2 - I_2^*}{I_2}\right)\left[\frac{\beta_2 S_2 I_1}{aS_2 + bI_1} - (m_2 + \mu_2) I_2\right].\end{aligned} \quad (57)$$

Let $S_1 = S_1 + S_1 - S_1^*$, $I_1 = I_1 - I_1^*$, $T = T - T^*$, $R = R - R^*$, $S_2 = S_2 - S_2^*$ and $I_2 = I_2 - I_2^*$. Then

$$\begin{aligned}\frac{dV}{dt}=&\left(\frac{S_1-S_1^*}{S_1}\right)\left[\pi_1-\frac{\beta_1\left(S_1-S_1^*\right)\left(I_2-I_2^*\right)}{a\left(S_1-S_1^*\right)+\in\left(I_2-I_2^*\right)}-\mu_1\left(S_1-S_1^*\right)\right]\\&+\left(\frac{I_1-I_1^*}{I_1}\right)\left[\frac{\beta_1\left(S_1-S_1^*\right)\left(I_2-I_2^*\right)}{a\left(S_1-S_1^*\right)+\in\left(I_2-I_2^*\right)}-\left(r_1+m_1+\mu_1+\gamma\right)\left(I_1-I_1^*\right)\right]\\&+\left(\frac{T-T^*}{T}\right)\left[r_1\left(I_1-I_1^*\right)-\left(\mu_1+\gamma\right)\left(T-T^*\right)\right]\\&+\left(\frac{R-R^*}{R}\right)\left[\gamma\left(I_1-I_1^*\right)+r_1\left(T-T^*\right)-\mu_1\left(R-R^*\right)\right]\\&+\left(\frac{S_2-S_2^*}{S_2}\right)\left[\pi_2-\frac{\beta_2\left(S_2-S_2^*\right)\left(I_1-I_1^*\right)}{a\left(S_2-S_2^*\right)+b\left(I_1-I_1^*\right)}-\mu_2\left(S_2-S_2^*\right)\right]\\&+I\left(\frac{I_2-I_2^*}{I_2}\right)\left[\frac{\beta_2\left(S_2-S_2^*\right)\left(I_1-I_1^*\right)}{a\left(S_2-S_2^*\right)+b\left(I_1-I_1^*\right)}-\left(m_2+\mu_2\right)\left(I_2-I_2^*\right)\right].\end{aligned}\tag{58}$$

Let P be equal to values of positive terms and Q be equal to values of negative terms of equation (58). Then after the expansion and collection of like terms together in the system we obtain

$$\frac{dV}{dt}=P-Q\tag{59}$$

where

$$\begin{aligned}P=&\frac{\left(S_1-S_1^*\right)\pi_1}{S_1}+\frac{\beta_1\left(S_1-S_1^*\right)\left(I_2-I_2^*\right)\left(I_1-I_1^*\right)}{I_1\left(a\left(S_1-S_1^*\right)+\in\left(I_2-I_2^*\right)\right)}+\frac{\left(T-T^*\right)\left(r_1\left(I_1-I_1^*\right)\right)}{T}\\&+\frac{\left(R-R^*\right)\left[\gamma\left(I_1-I_1^*\right)+r_1\left(T-T^*\right)\right]}{R}+\frac{\left(S_2-S_2^*\right)\pi_2}{S_2}\\&+\frac{\beta_2\left(S_2-S_2^*\right)\left(I_1-I_1^*\right)\left(I_2-I_2^*\right)}{I_2\left(a\left(S_2-S_2^*\right)+b\left(I_1-I_1^*\right)\right)},\end{aligned}\tag{60}$$

and

$$\begin{aligned}Q=&\frac{-\left(S_1-S_1^*\right)}{S_1}\left\{\frac{\beta_1\left(I_2-I_2^*\right)}{a\left(S_1-S_1^*\right)+\in\left(I_2-I_2^*\right)}+\mu_1\right\}-\frac{\left(I_1-I_1^*\right)^2\left(r_1+m_1+\mu_1+\gamma\right)}{I_1}\\&-\frac{\left(T-T^*\right)^2\left(\mu_1+\gamma\right)}{T}-\frac{\left(R-R^*\right)^2\mu_1}{R}\\&-\frac{\left(S_2-S_2^*\right)}{S_2}\left\{\frac{\beta_2\left(I_1-I_1^*\right)}{a\left(S_2-S_2^*\right)+b\left(I_1-I_1^*\right)}+\mu_2\right\}-\frac{\left(I_2-I_2^*\right)\left(m_2+\mu_2\right)}{I_2}.\end{aligned}\tag{61}$$

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Thus, from equations (60) and (61), if $P < Q$, then $\frac{dV}{dt}$ will be negative definite, meaning that $\frac{dV}{dt} < 0.$ It follows that $\frac{dV}{dt} = 0$ if and only if $S_1 = S_1^*$, $I_1 = I_1^*$, $T = T^*$, $R = R^*$, $S_2 = S_2^*$, $I_2 = I_2^*$. Therefore the largest compact invariant set in $\left\{\left(S_1^*, I_1^*, T^*, R^*, S_2^*, I_2^*\right) \in \Omega; \frac{dV}{dt} = 0\right\}$ is the singleton $\{E_1\}$ where E_1 is the endemic equilibrium of the model system (5). By LaSalles's invariance principle, then it implies that E_1 is globally asymptotically stable in Ω if $P < Q$.

4. NUMERICAL SIMULATIONS OF THE MODEL

In this section, we illustrate the analytical results of the study by carrying out numerical simulations of the model system (5) using a set of parameter values given in table 2. The values are obtained from published literature and other assumed parameter values with the initial conditions: $S_1(0) = 6500$, $I_1(0) = 40$, $S_2(0) = 15000$, $I_2(0) = 300$, $W(0) = 22$, $T(0) = 15.6^{\circ}C$.

Table 2: Model parameter Values and their interpretations to be used in the Numerical Simulations

Parameter	Meaning	Value	Source
π_1	Recruitment rate of susceptible humans	800	Chiyaka *et al.*, (2009)
π_2	Recruitment rate of susceptible snails	2500	Garira *et al.*, (2014)
μ_1	Human natural death rate	0.0000384	Feng *et al.*, (2002)
μ_2	Snail natural death rate	0.000569	Spear *et al.*,(2002)
m_1	Human death rate due to disease	0.0039	Feng *et al.*,(2004)
m_2	Snail death rate due to disease	0.0004012	Mangal *et al.*, (2008)
β_1	Disease transmission rate to the susceptible human by the infected snail in river	0.406	Spear *et al.*, (2002)
β_2	Disease transmission rate to the susceptible snail by the infected human in river	0.615	Mangal *et al.*,(2008)
r_1	Recovery rate of infected human due to treatment	0.49	Mushayabasa and Bhunu, (2010)
Q	Rate of ground water inflow to the river	$15000L^{-1}$	Assumed
c	Precipitation rate due to rainfall	0.155	Assumed
n	Rate of evaporation	0.66	Assumed
σ	Rate of environmental increase or decrease in temperature due to climate change	1.9999	Assumed
$T_{\max}$	Prescribed maximum environmental temperature increase $\left(50 - 1000^{\circ}C\right)$	$99^{\circ}C$	Assumed
$\in$	Rate of disease transmission by infected snail due to water level	0.0002	Spear *et al.*,(2002)
b	Rate of disease transmission by infected human due to water level	0.00022	Assumed
a	Impact of water level on disease transmission	0.00025	Assumed

Figures 2 show the dynamics of state variables for human population. Figure 2(a) shows the susceptible human while figure 2(b) shows the infected human in the population.

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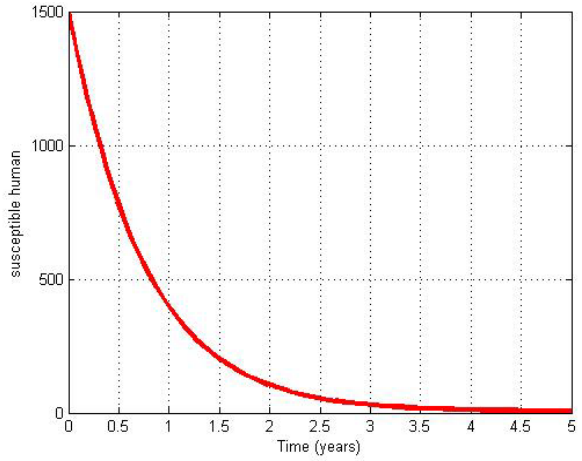

Figure 2(a)

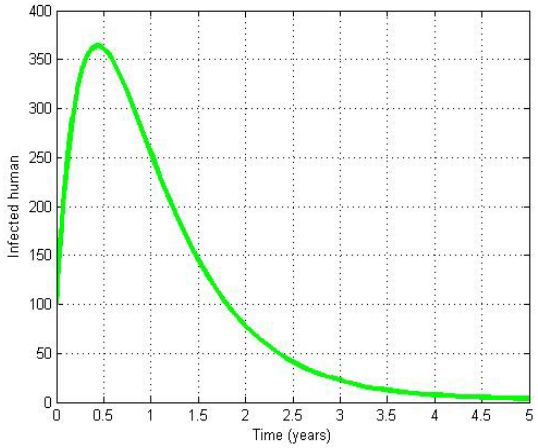

Figure 2(b)

Figures 2: Dynamics of the State Variables of the Schistosomiasis Dynamics on (a) susceptible human and on (b) infected human in the population

From figure 2(a), it is observed that the number of susceptible individuals in the population decreases with time. This happens due to susceptible humans who are exposed to infection, die naturally. Similarly, figure 2(b) shows that infected humans increase due to higher number of susceptible humans moving from susceptible class to infected class in the population and then decrease exponentially time due to schistosomiasis-induced deaths, natural deaths and those who recover from treatment which is practiced in the community.

Figures 3 show the dynamics of susceptible snails and infected snails.

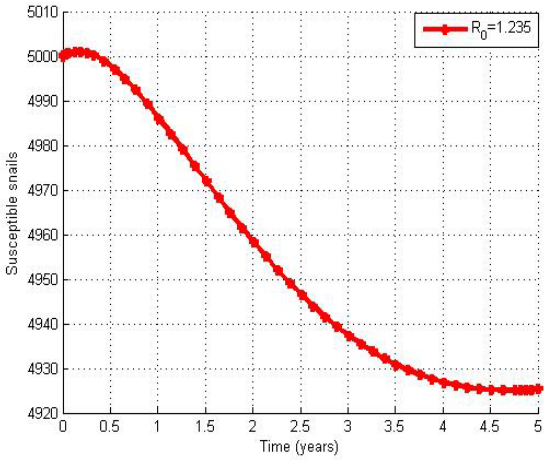

Figure 3(a)

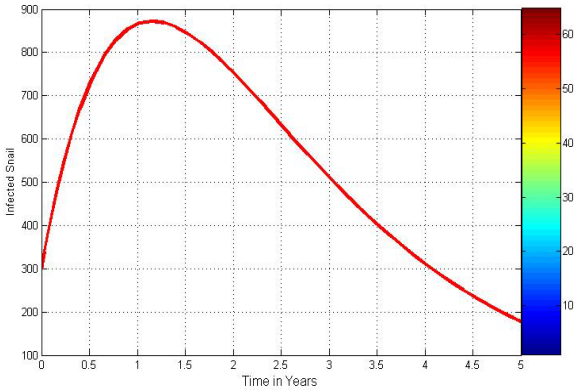

Figure 3(b)

Figure 3: State Variables of the Schistosomiasis Dynamics on (a) Susceptible snails and (b) Infected Snails in the Population

From the two graphs it is seen that the susceptible snails in figure 3(a) are decreasing with time. This is due to the fact that some of them are dying naturally while others are moving to the infected class after being infected by infested human wastes in the river. Similarly, in figure 3(b) it is observed that there is an exponential increase of infected snails up to a certain level and eventually started decreasing with time. The exponential increase of infected snails is due to high number of susceptible snails in the population being infected, also the exponential decrease of infected snails with time is due to disease-induced deaths and natural death of infected snails.

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Figures 4 show the rate of environmental increase in temperature due to climate changes and the rate of environmental decrease in temperature due to climate changes respectively.

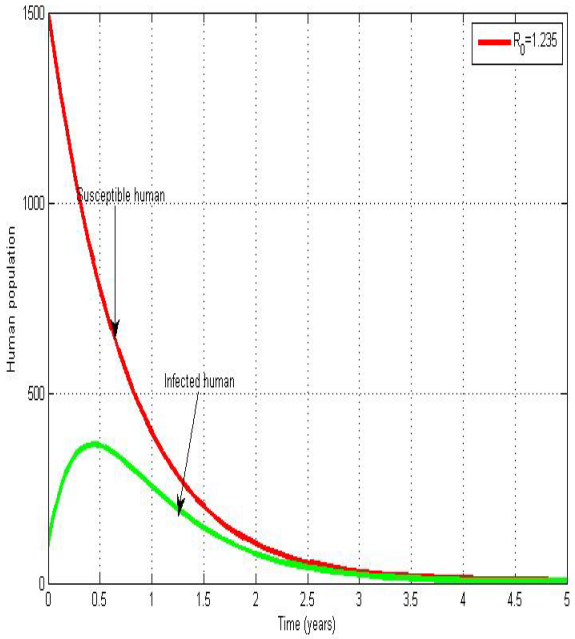

Figure 4(a)

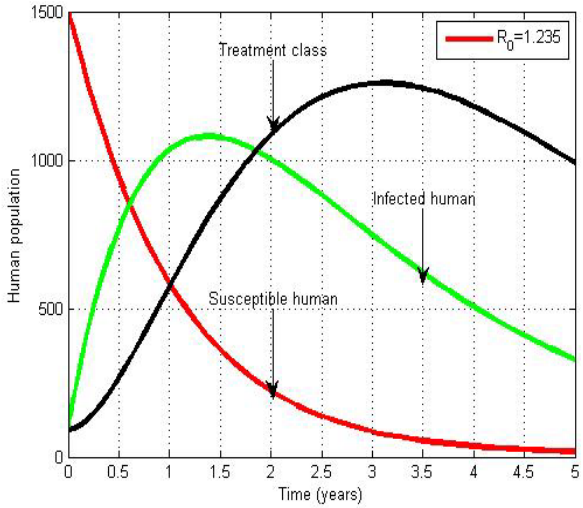

Figure 4(b)

Figure 4: Results showing the increase in temperature and the decrease in temperature due to climate changes

From the figure 4(a) it is observed that as the number of susceptible humans decreases exponentially due to infection. The number of infected population is found to be increasing at a certain time interval and then start decreasing with increase of time. The other reasons for susceptible class to decrease with increase of time may be is due to natural death, and on the other hand the infected class of humans is decreasing perhaps is due to disease-induce rate and natural death in the particular population. On the other hand, figure 4(b) shows an addition of treated class. It is seen that the graph of treated individuals is increasing after the application of medicine such as praziquantel to the infected population.

Figures 5 show the evolution with time of infected, treated and recovered population.

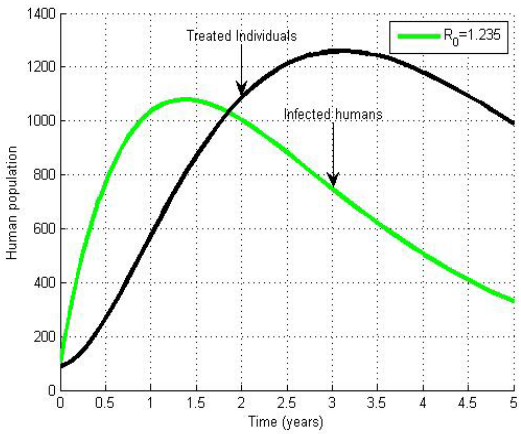

Figure 5(a)

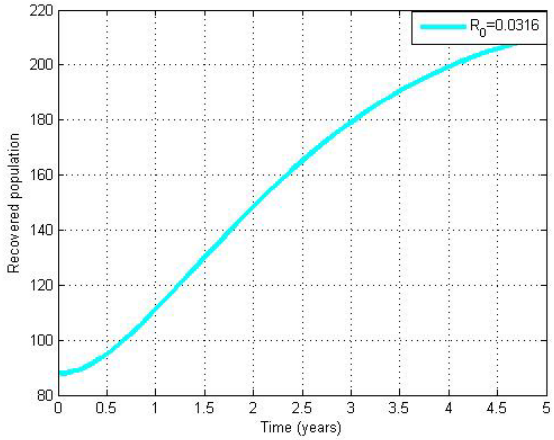

Figure 5(b)

Figures 5: Results Showing the Evolution of Infected, Treated and Recovered Population with time

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The simulation results shown by figure 5(a) shows that the high amount of cercariae released by infected snails to the physical water environment causes the higher rate of infection at an individual level. The number of infected individuals will continue to increase until proper measurements are taken to combat the disease. The decreasing graph of infected groups with increase of time indicates that some individuals are recovering due to treatments in the community as shown by treatment graph in the same figure 5(a). Natural deaths and disease induce death rates are among of the reasons which causes the decrease of infected population. On the other hand, figure 5(b) shows the recovered population after proper treatments are taken in the particular population of infected individuals. Thus treatment has a positive impact to the society.

Figures 6 show the numerical solutions of infected, treated and recovered individuals in the population.

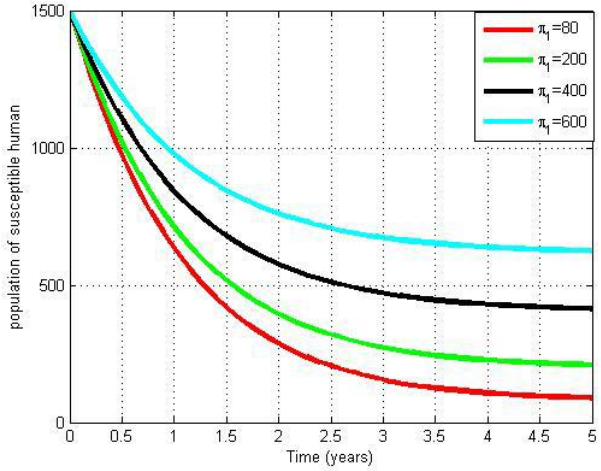

Figure 6(a)

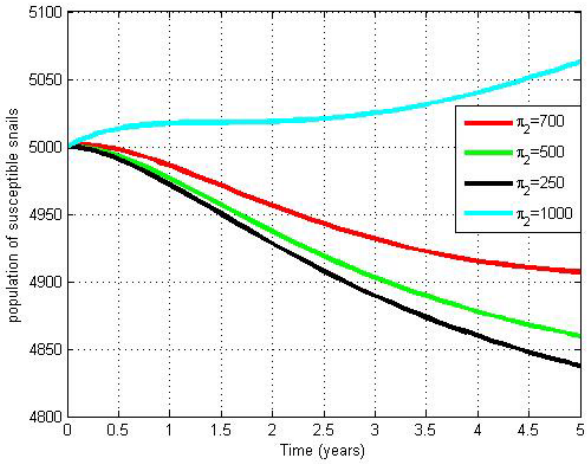

Figure 6(b)

Figure 6: Results showing the rate of recruitment for both susceptible human and snails at a particular population

Figures 6 illustrate the graphs of numerical solutions showing propagation of susceptible humans and susceptible snails for different values of rate of recruitments. The rate of recruitment for new susceptible humans are $\pi_1 = 80,\ 200,\ 400,\ 600$ and the rate of recruitment for a new susceptible snails are $\pi_2 = 250,\ 500,\ 700$ and 1000 respectively. We note from figure 6(a) that the emerging of new susceptible humans increase the intensity of human population at an individual level, also from figure 6(b), it is observed that the new susceptible snails increase the intensity of snail population in the physical water environment.

We note that when the rate of recruitment for susceptible snails is high in the physical water environment and when the rate of miracidia released by human wastes increases in the physical water environment lead to the higher number of snails infection rates.

The higher the number of infection rate of snails leads to a higher number of cercariae in the physical water environment, which upon when in contact with humans accelerates the rate of re-infections again and a big population will likely suffer form the disease. It is suggested that elimination of snails by the application of molluscicides is the best solution to reduce or eliminate the disease.

Figures 7 show the natural death rate of susceptible humans and susceptible snails in the physical water environment.

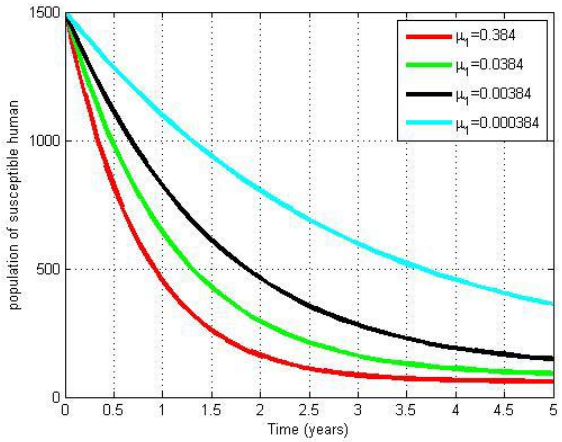

Figure 7(a)

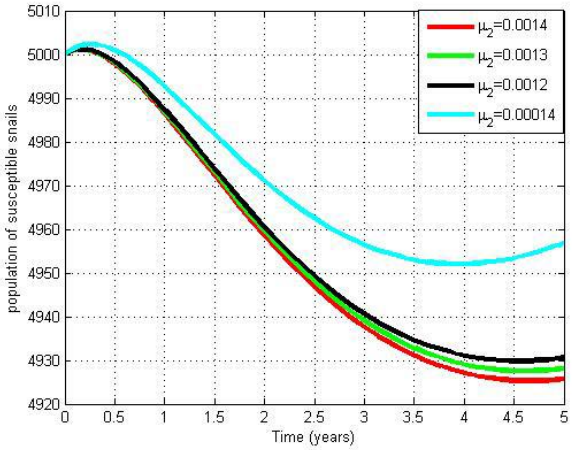

Figure 7(b)

Figures 7: Effects of Variations of River Water Levels on Schistosomiasis Epidemic in Snails

From the figures 7, it is observed that when the rate of natural death is low, the number of susceptible humans and snails are higher in the entire population as indicated by $\mu_1 = 0.000384$ and $\mu_2 = 0.000014$ respectively. As the natural death rate increase for both populations, the number of susceptible humans is found to be decreasing at a higher rate as indicated by $\mu_1 = 0.384$. Likewise, the number of susceptible snails in the physical water environment are also removed fast as indicated by $\mu_2 = 0.0014$. It is also noted that the death rate of snails reduce the intensity of disease at individual level, so it is suggested that an elimination of snails which act as an intermediate host in the disease transmission in all sources of water bodies will help to reduce schistosomiasis in the community.

Figures 8 show different rates of disease transmission for infected individuals in the population and disease induce death rates.

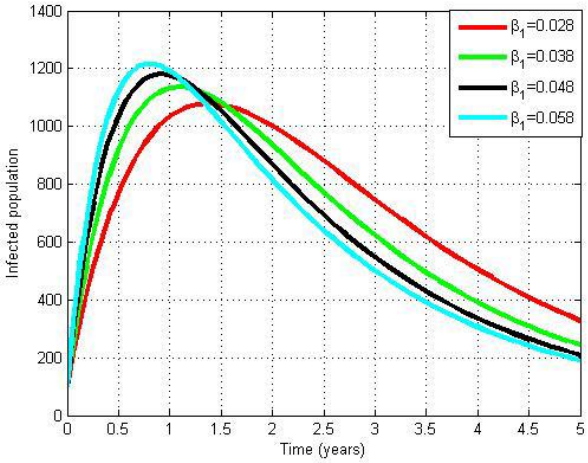

Figure 8(a)

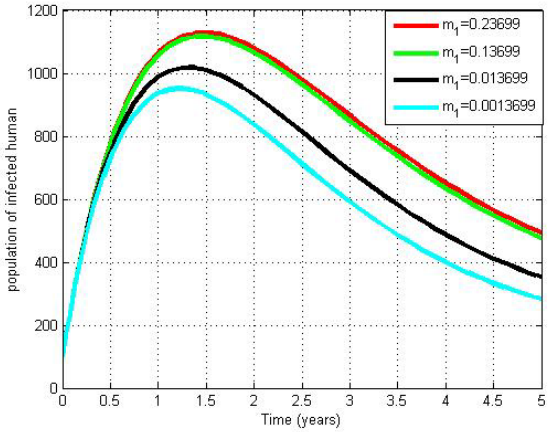

Figure 8(b)

Figures 8: Dynamics of Infected Humans under Different Rates of Disease Transmission, β_1 and Disease Induce Death Rate m_1

From figure 8(a), it is observed that the rate of disease transmission to the susceptible human by cercariae released by infected snails in the physical water environment depends on the ambient temperature. When the ambient temperature is below a certain level which do not favour the reproduction of snails, the rate of transmission is also found to be low as shown by $\beta_1 = 0.028$ and $m_1 = 0.038$ above, but when the ambient temperature increases up to a certain level where it become conducive for snails reproduction, we observe that the rate of disease transmission increases. This happens due to the fact that more snails get recruited in the physical water environment and more snails get infected by miracidia which results in a large amount of cercariae parasites which again infects human population at a higher rate as illustrated by $\beta_1 = 0.058$ in the figure 8(a) above.

The high number of human infection rate increases the death rate due to disease for infected individuals in the population as shown by figure 8(b). When the infection is high, the death due to disease is also found to be high as indicated by $m_1 = 0.23699$ and $m_1 = 0.13699$. As the rate of transmission decreases with time, it is observed that the number of deaths decreases as shown by $m_1 = 0.0013699$ in figure 8(b). It is suggested that human behaviour changes which reduce contact with infected water bodies and good sanitation should be practiced in the community.

Figure 9 shows the dynamics of infected snails under different rates of disease transmission β_2.

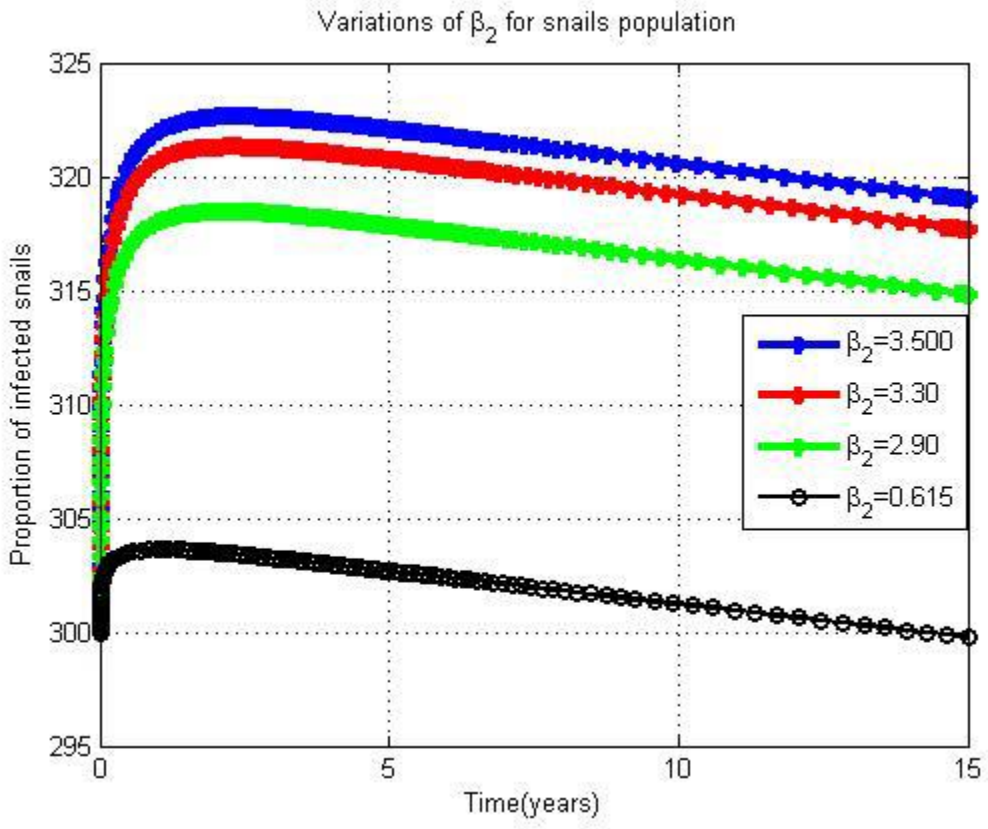

Figure 9: Different rates of disease transmission for infected snails in the population

Figure 9 illustrates graphs of numerical solutions showing different rates of disease transmission to the susceptible snails by the infected humans waste in the physical water environment. The rate of snails infection is high when the amount of infected human wastes is also large in the physical water environment, and this is shown by $\beta_2 = 3.50$ from the given figure above. As the amount of infected waste decreases, it is also observed that the rate of snails infection decreases with increase of time, as seen in the figure above where $\beta_2 = 3.30$, $\beta_2 = 2.90$ and $\beta_2 = 0.6150$. Again the transmission of the disease from humans to snails depends on the ambient temperature of the environment. The values for different rates of disease transmission to the susceptible snails by infected human waste in river are as follows: $\beta_2 = 3.500$, $\beta_2 = 3.300$, $\beta_2 = 2.900$, $\beta_2 = 0.615$.

Figure 10 shows different values of disease transmission by infected humans due to river water levels b.

Figure 10 shows the graphs of numerical solutions showing different values of disease transmission by infected human due to river water levels. When the levels of river water is low, the rate of disease transmission become high if that amount of water in infested by cercariae as it is shown by $b = 0.0002$ in the figure 10. As the levels of river water increases, the force of infection to susceptible individuals decreases which leads to the decrease of infection rates for human in the population as shown by $b = 0.02$ and $b = 0.2$ respectively. The tested values for rates of disease transmission by infected humans due to infested river water levels by cercariae are: $b = 0.0002$, $b = 0.002$, $b = 0.02$, $b = 0.2$.

Figure 11 shows different values for disease transmission by infected snails due to water levels, $\in$.

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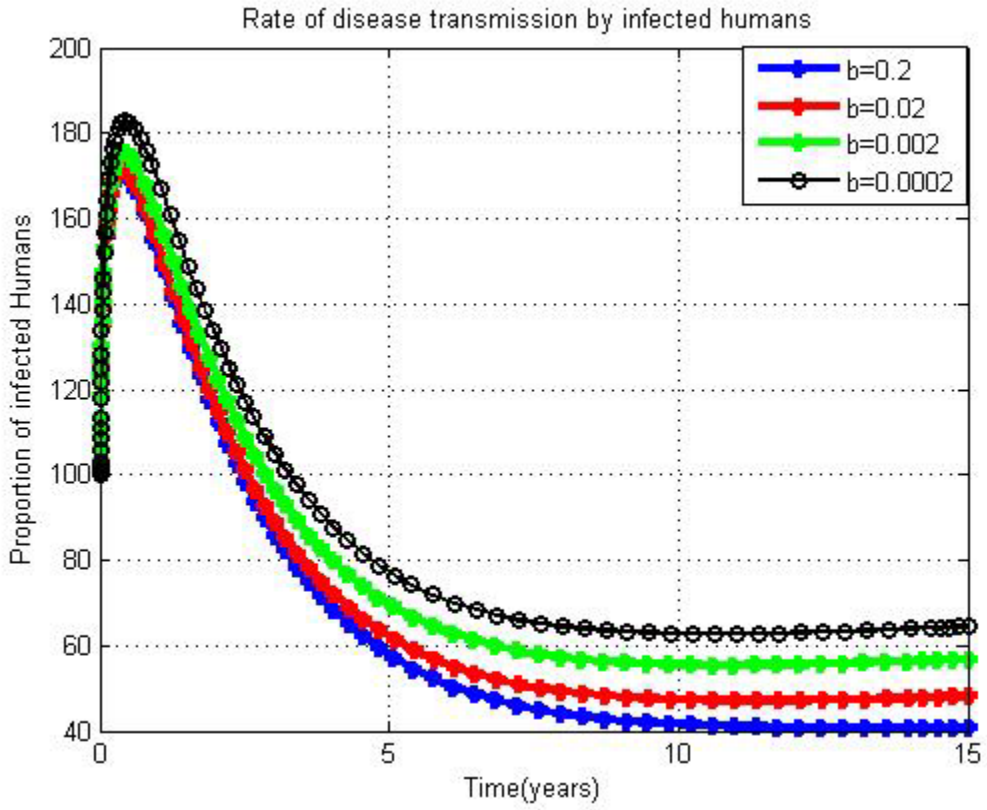

Figure 10: Different values of disease transmission by infected human due to water levels

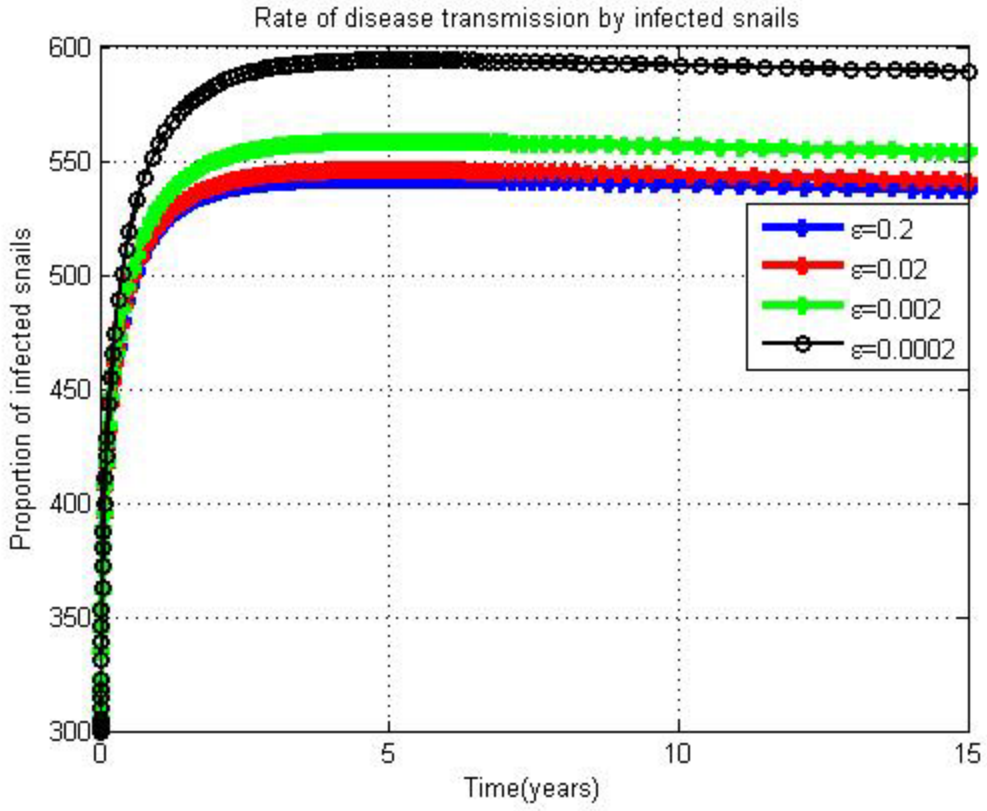

Figure 11: different values for disease transmission by infected snails due to water levels

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From the figure 11, it is observed that number of infected snails which is presented by $\in = 0.0002$ is high only when the levels of infected river water by miracidia is low, say $\in = 0.0002$, as the levels of infected river water increases say from $\in = 0.0002$, to $\in = 0.002$, the number of infected snails are also found to be decreasing with time as seen when $\in = 0.002$. This trend is expected to continue until there is no further infection when the levels of river water is maximum. Values for different variations of river water levels used in the simulation are: $\in = 0.0002$, $\in = 0.002$, $\in = 0.02$ and $\in = 0.2.$

Figure 12 shows the recovery rate of infected humans due to different rates of treatments r_1.

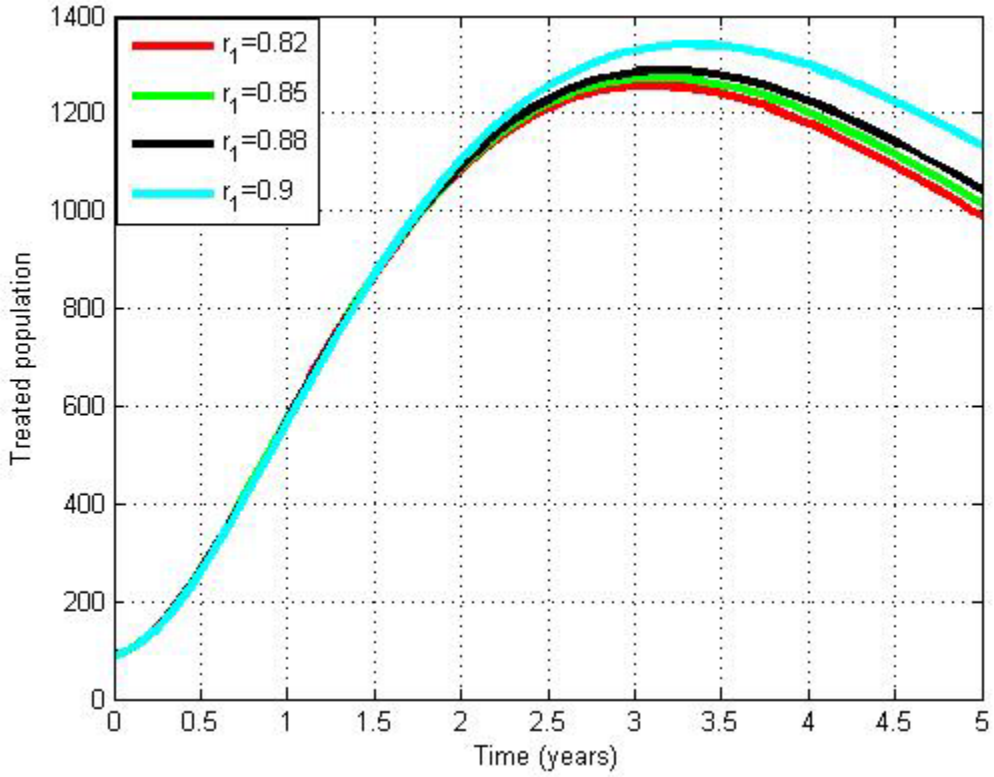

Figure 12: Recovery rates of infected human due to different rates of treatments

From the figure 12, it is observed that the application of treatments to the infected individuals in the population is an important factor in the elimination of the disease. Figure 12, presents recovery rates of infected population by the application of medicine. $r_1 = 0.82$ indicates low amount of medicine supplied to treat the infected people in the community, but as you increase the rate of treatments in the population, all infected individuals will start to recover at different rates depending on the rates of treatments applied as shown above. $r_1 = 0.9$ indicates higher rate of recovery because of proper and efficient treatments conducted in the community.

Figures 13 show the variation of natural recovery and recovery rate due to treatment by infected population.

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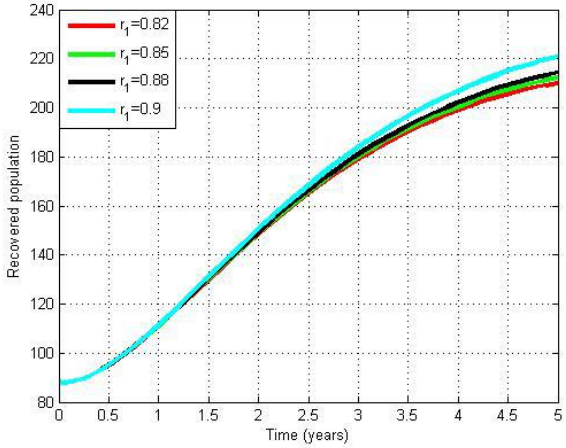

Figure 13(a)

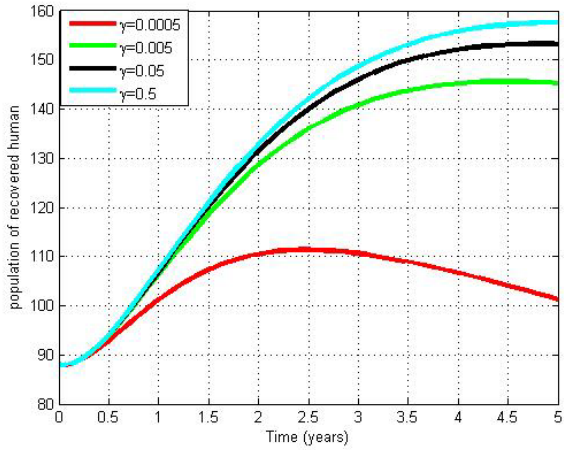

Figure 13(b)

Figures 13: Results showing the comparison of natural recovery and recovery due to treatments to humans population

From figure 13, it is seen that the rate of natural recovery of infected individuals depends on the immunity within an individual. If the immunity is strong within an infected human, the rate of natural recovery also become fast as indicated by figure 13(b) for $\gamma = 0.5$ and $\gamma = 0.05$, but the lower the immunity leads to slow natural recovery of individuals as demonstrated by γ=0.0005. On the other hand the application of strong and proper medicine within an infected individuals helps to cure the disease and recover at a higher rate as indicated by figure 13(a) for $r_1 = 0.9$.

5. CONCLUSIONS AND RECOMMENDATIONS

In this study, we have formulated and analysed a mathematical model of schistosomiasis concerning the impact of climate changes on the disease transmission in human. Temperature variation and the different levels of river water were considered as agents of climate changes on the disease transmission. The dynamics of human population and snails population have been incorporated with environmental factors such as the effect of climatological environment. Both qualitative and numerical analysis of the model have been used to determine the basic reproduction number as well as the existence and stabilities of the model equilibrium points. Qualitative analysis showed that the model has two equilibria, namely: the disease free equilibrium point and endemic equilibrium point. Using next generation method, it was found that the disease free equilibrium is locally asymptotic stable when the reproduction number $R_0 < 0$, and unstable otherwise. In the case of endemic equilibrium point, the qualitative analysis was done by using Centre manifold theorem developed by Castillo-Chaves and Song (2004). It was concluded that near the threshold $R_0 = 0$, there exists a stable endemic equilibrium point which is globally asymptotically stable if $R_0 > 0$.

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Numerical simulations were performed and it was observed that the transmission of schistosomiasis in both human and snails is effective when environmental temperature favours the reproduction of snails. It was also noted that when the temperature was below a certain level, the rate of infection was observed to be decreasing as a results of snails been in the hibernation state. Likewise, when the environmental temperature is above a certain level, it was noted that the rate of infection was also decreasing, due to the fact that all infected snails become unable to release or shed cercariae to the physical water environment. Furthermore, simulation results showed that variation of river water levels where miracidia, cercariae and snails live, had an effect on the disease transmission. It was observed that when the levels of river water is low the number of infected humans by schistosomiasis in the population is larger than when the levels of river water is high.

Eradication of schistosomiasis remains a challenge in most of the developing countries in the world, hence there is a need to strengthen the control strategies at hand as well as putting more efforts to overcome the epidemic especially to those areas close to rivers, dams and ponds. Thus, it is recommended that:

1. Human behavioural changes which reduce contact with infected water bodies should be practiced so as to reduce the infection intensity at individual levels,
2. Good sanitation practices should be applied by the community which reduce contamination of water bodies so as to reduce the rate of infection in humans at individual level,
3. Elimination of snails population using molluscides should be practiced by public health interventions,
4. More awareness campaigns on the side-effects of schistosomiasis epidemic to humans, should be practiced especially to those areas close to waterbodies.

Conflict of interest

The authors declare that they have no conflict of interest.

Funding:

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Ethical approval

This article does not contain any studies with human participants performed by any of the authors.

Data and materials availability:

All data associated with this study are present in the paper.

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